

SUDECK'S BONE ATROPHY
CLINICAL AND EXPERIMENTAL FINDINGS

BY
DONALD SIDNEY MILLER

B. S., University of Illinois, 1930

M. S., University of Illinois, 1932

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AN ABSTRACT OF A THESIS
SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS
FOR THE DEGREE OF DOCTOR OF PHILOSOPHY IN
SURGERY IN THE GRADUATE SCHOOL OF THE
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The subject of post-traumatic, painful osteoporosis, or Sudeck's bone atrophy has been a frequent source of argument, particularly, regarding the etiology of this condition and its treatment. The various etiological factors which have been discussed in most of the articles written on this subject have been the following: Trophoneurosis theory, Non use theory, Hyperemia theory, and the Infectious theory.

In the work herein discussed, attempt has been made to ascertain, by plethysmographic methods, the actual status of the blood supply to the involved extremity. These results so attained, will be interpreted physiologically. Associated with the above work in clinical patients, attempts to produce bone atrophy were made in animals (dogs). The methods used in the latter, to produce atrophy of bone, were the following: sympathetic ganglion and nerve stimulation, sympathectomy (with and without the application of casts) ligation of both artery and vein of the hind limb of the animal, stimulation of nerve trunks, and surgery on the osseous structure of the hind limb of the animal.

Sudeck, in 1900, first wrote on this condition and described a symptom complex consisting of an acute onset with pain in the joint following trauma, osteoporosis of the involved joint, and trophic disturbances limited to the area of injury. He concluded that this condition was an inflammatory irritation of the bone which, in turn, led to bone absorption. Kienboch, in 1902, suggested that this condition was due to a trophoneuritic element which existed following injury. Since this period, a great deal of work has been done on the subject, but mostly of a clinical nature. The most important controversy that has existed concerns the status of the blood supply to the involved extremity. Brandes (1913) did not believe that Sudeck's bone atrophy, so described, existed, while Hilgenreiner (1923) believed that this entire condition may be attributed to non-use. Allison and Brooks (1921) and Gray and Carr (1915) were the earliest of the American authors attempting to produce acute bone atrophy experimentally. Most of the previous work has been reported in European literature.

CLINICAL AND EXPERIMENTAL FINDINGS

The clinical study of this condition involved twenty patients with Sudeck's bone atrophy. This entire group belonged to the post-traumatic type. Plethysmographic tracings were made on twelve of this group. This method was used because it gave a direct measure of blood flow to the part involved. The normal extremity was used as a control. Each patient was studied over a period of many months, receiving from five to twenty tracings at each sitting. In several patients, blood flow tracings were made in response to heat and cold environment; this was done by changing the temperature of the water surrounding the limb.

The end results of blood flow measurements in the twelve patients indicated a consistent elevation in the involved extremity as compared to the normal one. Responses to heat and cold indicated that the involved extremity had a more stable type of circulation than that found in the normal control member, i.e., the blood flow in the involved extremity did not fluctuate as much under the cold and under the heat as did the normal extremity. Aside from plethysmographic blood flow tracings, the patients also were subjected to histamine reaction flares, oscillometric recordings, and studies in the blood calcium, phosphorus and phosphatase. In the majority of cases there was an increase in the phosphatase content of the blood. In both the histamine and oscillometric readings, the results indicated that the circulation was greater in the involved extremity as compared to the normal extremity. Four patients had sympathectomies for this condition. All were relieved to a great extent of their pain. Weight bearing was not as painful, and a subsequent re-calcification of bone took place following several months.

The experimental methods used to produce bone atrophy in dogs, included sympathetic irritation to the chain by various chemicals, sympathectomy (with and without cast application), blood vessel ligation, nerve irritation, and injury to the osseous structure of the extremity. It was found that the most consistent amount of atrophy of the bone in any single group of experiments resulted from sympathectomy with the application of a

cast on the side of the operation. It was also found that any method of stimulation, whether it be to nerve, muscle, bone or blood vessel produced a small degree of atrophy of bone. X-Ray studies were made every two to three weeks following surgery, a control being taken of the opposite extremity with the same X-Ray technique.

SUMMARY AND CONCLUSIONS

A modified method of measuring blood flow has been used to determine the status of circulation in Sudeck's bone atrophy. Twelve patients over a period of a year and a half to two were studied by the Author's plethysmographic machine. Studies of the blood chemistry, histamine flare and oscillometric tests were also made and recorded. Responses of the extremity, both the involved and normal, to heat and cold environment, were also recorded. The results from sympathectomy were good and are here suggested as the method of treatment in Sudeck's bone atrophy. Various attempts to produce bone atrophy in dogs, as above described, have also been reported elsewhere.

CONCLUSIONS:

1. Sudeck's atrophy, or post-traumatic osteoporosis is a real clinical entity.
2. The disease is more common than supposed and is the cause of a chronic, intractable type of pain in an extremity producing a marked disability following the injury.
3. There is a definite psychoneurotic element often present in patients afflicted by this dystrophy. This is more evident in the severe, intractable cases, perhaps as a result of unrelieved pain.
4. The earlier the bone atrophy appears and the more severe the atrophy is, the more disability exists and the more difficult it is to treat.
5. Pain, vasomotor disturbances, and osteoporosis are the three most important elements in this condition, following trauma.
6. From experimental findings, it is possible to produce a type of atrophy in dogs that is similar in appearance roentgenologically but not clinically, to that of Sudeck's atrophy. This was

seen when sympathectomy was done and immobilization produced, and in animals having had nerves stimulated together with irritation of articular structures.

7. The percentage of bone atrophy following trauma is not great.

8. There is a vasodilatation produced following all trauma which is due, in all likelihood, to some local chemical substance produced by way of an axone reflex.

9. The possible mechanism of the production of this clinical entity is an exaggerated response of the vegetative nervous mechanism to trauma.

10. The increase in blood flow following exposure to heat is less marked in the Sudeck extremity than on the normal limb. The production of blood flow following application of cold in the Sudeck extremity is less than the reduction of blood flow in the normal extremity under the same circumstances.

11. The blood phosphatase is increased in the majority of cases.

12. The association between Sudeck's bone atrophy and senile atrophy, the former being a stimulus to the latter, has been suggested.

13. Sympathectomy is the most effective type of treatment in Sudeck's atrophy; repeated sympathetic block may be tried first. Sympathectomy does not interrupt any of the affected pathways, but may act by "washing out" the pain substance or by abolishing capillary dilatation, as sympathetic fibers carry capillary dilators.

14. A modification of the Abramson plethysmographic machine has been used in the above experiments and has proved to be a direct and efficient means of determining blood flow.

VITA

Donald Sidney Miller

Born ni Maywood, Illinois, December 9, 1908; attended the public schools in Chicago and graduated from the Cregier grammar school in 1922 and from the McKinley high school in 1925. Attended the Crane college, Loyola Dental college, and had pre-medical training at the University of Illinois in Champaign. Received a B.S. degree at University of Illinois Medical School in 1930; M.S. and M.D. degrees at the University of Illinois College of Medicine in 1932. Elected into Alpha Omega Alpha honorary society in 1931 and into Sigma XI in 1941; graduate student in surgery from 1937 to date.

Interned at the Cook County Hospital 1933-1934; resident at Cook County Hospital 1934-1937; post-graduate work in Bologna, Italy and Vienna (six months). Teaching staff of the Cook County Graduate School, member of Chicago Medical Society, Chicago Orthopedic Society, American College of Surgeons, American Academy of Orthopedic Surgeons, member of the American Board of Orthopedic Surgery, and member of the American Association of Industrial Physicians and Surgeons. Associate surgeon in Orthopedics, Cook County Hospital; attending staff, Oak Park Hospital.

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